



Contents lists available at ScienceDirect

International Journal of Psychophysiology

journal homepage: www.elsevier.com/locate/ijpsycho

REM sleep as a potential indicator of hyperarousal in psychophysiological and paradoxical insomnia sufferers ☆, ☆, ☆

Alexandra D. Pérusse*, Maude Pedneault-Drolet, Christine Rancourt, Isabelle Turcotte, Geneviève St-Jean, Célyne H. Bastien

École de psychologie, 2325 rue des Bibliothèques, Université Laval, Québec, QC, G1V 0A6, Canada

Laboratoire de sommeil et potentiels évoqués cognitifs, 2525 chemin de la Canadière, Centre de recherche de l'Institut universitaire en santé mentale de Québec, Québec, QC, G1J 2G2, Canada

ARTICLE INFO

Article history:

Received 6 September 2014

Received in revised form 30 November 2014

Accepted 9 January 2015

Available online xxxx

Keywords:

Insomnia

REM sleep

Hyperarousal

Macrostructure

Microstructure

Arousals

ABSTRACT

Study objectives: The objective was to study REM sleep macrostructure and microstructure as potential indicators of hyperarousal in insomnia by comparing good sleepers (GS) and insomnia sufferers (INS) (subdivided into psychophysiological "PSY-I" and paradoxical "PARA-I").

Design: Cross-sectional comparisons of GS, PSY-I and PARA-I.

Setting: Participants slept for 4 consecutive nights in the laboratory where PSG was recorded. Nights 2 and 3 were combined to compare REM sleep between groups.

Participants: Thirty-nine PSY-I, 27 PARA-I and 47 GS completed the study, comprising home questionnaires, clinical interviews and night PSG recordings. All participants were aged between 25 and 55 and met inclusion criteria for either PSY-I, PARA-I or GS.

Interventions: N/A.

Measurements and results: Results showed no between group differences on REM sleep macrostructure. As for REM sleep microstructure, PSY-I had an increased number of wake intrusions compared to PARA-I ($p = .03$). Subjective SE, TST and TWT were significantly correlated with the duration of REM sleep (REMD; $p \leq .002$) and with the proportion of REM sleep for PARA-I ($p \leq .06$).

Conclusions: REM sleep macrostructure does not seem to be an adequate indicator of hyperarousal in insomnia. However, the number of wake intrusions in REM could be used to differentiate PSY-I from PARA-I and could reflect the heightened arousal of the former group. Relationships between REM sleep duration and proportion could be linked to dream imagery activity, especially in PARA-I. Further investigations are needed to identify variables that could reflect hyperarousal and differentiate insomnia types.

© 2015 Elsevier B.V. All rights reserved.

1. Introduction

Insomnia is one of the most common sleep disorders reported in the general population; more than 13% suffering from chronic insomnia (Morin et al., 2011) and between 30 and 48% occasionally reporting insomnia related symptoms (Ohayon, 2002). Insomnia can be divided into different types; psychophysiological insomnia (PSY-I) and paradoxical insomnia (PARA-I) being the most prevalent (AASM, 2005). PARA-I is characterized by misperceptions of sleep quality and quantity.

Individuals suffering from PARA-I report important sleep difficulties which are hardly documented with basic objective sleep measures (polysomnography; PSG; Edinger et al., 2004). However, they complain of diurnal symptoms, to the same extent as other types of chronic insomnia sufferers. On the other hand, PSY-I is characterized by "relatively" good perceptions of sleep duration and quality along with objective sleep difficulties (for a review see Bastien, 2011). The maintenance of PSY-I results from the conditioning between sleep related stimuli (e.g. bedroom) and anxious thoughts concerning possible sleep disturbances (Espie, 2002; Harvey, 2002). This conditioning contributes to the elevated cognitive activation typically reported in insomnia sufferers (INS; Wicklow and Espie, 2000).

Diagnostically, insomnia is characterized by sleep onset and/or sleep maintenance difficulties and/or early morning awakenings (AASM, 2014; APA, 2013). In addition to these sleep difficulties, INS often experience important daytime repercussions such as fatigue, diurnal sleepiness, confusion, sudden mood changes and cognitive alterations (Ohayon, 2002). These consequences suggest that insomnia is a 24 hour problem, a concept being increasingly supported by empirical studies, especially

☆ This study was supported by the Canadian Institutes of Health Research (CIHR; # 49500, 86571).

☆☆ Conflict of interest statement: The authors have no potential conflict of interest to declare.

* Corresponding author at: Pavillon Félix-Antoine-Savard, 2325 rue des Bibliothèques, Bureau 1010, Université Laval, Québec, QC, G1V 0A6, Canada. Tel.: +1 418 656 2131x11316.

E-mail addresses: alexandra.duchesne-perusse.1@ulaval.ca (A.D. Pérusse), maude.pedneault-drolet.1@ulaval.ca (M. Pedneault-Drolet), christine.rancourt.2@ulaval.ca (C. Rancourt), isabelle.turcotte.2@ulaval.ca (I. Turcotte), stjeang@csdraveurs.qc.ca (G. St-Jean), celyne.bastien@psy.ulaval.ca (C.H. Bastien).

in the hyperarousal domain (Bonnet and Arand, 1995, 1998, 2000; Edinger et al., 2008; Péresse et al., 2013; Roehrs et al., 2011; Stepanski et al., 1988). In the context of insomnia, hyperarousal can be defined as the elevation of somatic, cognitive and cortical activations. Hyperarousal is one of the core features of this sleep disorder, as suggested by the neurocognitive model of insomnia (Perlis et al., 1997). In this model, the authors state that INS, so to palliate for their sleep difficulties, tend to develop maladaptive behaviors such as increasing time spent in bed and going to bed earlier. These strategies are not efficient since they contribute to the elevation of somatic, cognitive and cortical activations (Morin, 1993). In the neurocognitive model (Perlis et al., 1997), the relationship between insomnia and cortical hyperarousal was established by focusing on some aspects of sleep microstructure, such as electroencephalography (EEG) frequencies, while limited attention was conducted towards sleep macrostructure. In addition, sleep stages were not distinguished, contributing to a difficulty in establishing the consequences of hyperarousal throughout the night in insomnia.

This field of research has evolved and, by distinguishing sleep stages, hyperarousal could be reflected in sleep macrostructure. The influence of hyperarousal on the macrostructure of non-rapid eye movement (NREM) sleep was the principal interest, providing empirical evidences for the neurocognitive model (Bastien et al., 2003; Parrino et al., 2009; Okura et al., 2008; Thacher et al., 2006). On the other hand, impacts of insomnia on the macrostructure of rapid-eye movement (REM) sleep were seldom investigated nor linked to hyperarousal. In general, INS' nights were characterized by a significantly smaller proportion of REM sleep compared to good sleepers (GS; Bonnet and Arand, 1995; Feige et al., 2008; Jurysta et al., 2009; Merica et al., 1998; Nissen et al., 2011; Voderholzer et al., 2003). Nonetheless, opposite results were also observed, thus that INS spent more time in REM sleep than GS (Lamarche and Ogilvie, 1997; Okura et al., 2008) or no differences at all (Hairston et al., 2010). Altogether, results tend to imply a lower proportion of REM sleep in INS compared to GS (Baglioni et al., 2013). Results for the length and latency of REM periods are quasi-inexistent. While Merica et al. (1998) observed shorter periods of REM sleep in INS, Feige et al. (2013) failed to find differences in latency of REM periods between INS and GS.

Studies on the microstructure of REM sleep (wake intrusions, arousals and eye movements) are even scarcer than those on the macrostructure. Feige et al. (2008) studied wake intrusions during REM sleep as well as arousals and eye movements (EMs) in INS. They reported a reduced number of EMs in INS during REM sleep. However, the densities of EMs in INS were not significantly lower, suggesting that the reduction in EMs probably resulted from a reduced duration of REM sleep (Feige et al., 2008). Finally, in the same study, the amount of arousals and wake intrusions was significantly higher in INS compared to GS, suggesting that REM sleep of the former group was more fragmented and consequently, reflected an increased state of arousal in INS. Feige et al. (2008) also showed that the amount of REM sleep contributed to subjective wake time in INS. The same group of authors (Riemann et al., 2012) later postulated that specific hyperarousal systems might be targeted in REM sleep of INS and would add to the misperception of sleep, an hypothesis also set forward by Bastien et al. (2013).

Thus far, the relationship between hyperarousal in REM sleep and insomnia types has been seldom studied. Our group showed, using power spectral analysis (PSA), lower relative powers in delta, theta and alpha bands in PARA-I's REM sleep compared to PSY-I and GS, indicating a higher cortical activation in the former group (St-Jean et al., 2013). That same year, deficits in inhibition processes during REM sleep, as studied with event-related potentials (ERP), were more likely to appear in PARA-I than in PSY-I (Bastien et al., 2013). Therefore, it might be that hyperarousal is channelled through a different aspect of information processing between PARA-I and PSY-I. Since differences related to cortical hyperarousal levels have been observed between PARA-I and PSY-I during REM sleep, the classification of insomnia types is justified

when hyperarousal is studied. However, no studies addressing the differences of REM sleep macrostructure and microstructure (arousals, wake intrusions and EMs) between PSY-I and PARA-I has been conducted yet. This clustering would provide a more representative understanding of hyperarousal in insomnia and how it is reflected in the macrostructure and the microstructure of REM sleep of suffering individuals.

1.1. Objectives and hypotheses

This study has for main objective to study hyperarousal in REM sleep, and more specifically, if both REM macrostructure and microstructure can be recognized as potential indicators of hyperarousal in INS and two of its types, psychophysiological (PSY-I) and paradoxical (PARA-I).

1.1.1. Macrostructure

We hypothesize that REM sleep macrostructure will reflect the hyperarousal of INS, especially in PARA-I, who grossly misperceive sleep. We predict that the proportion of REM sleep, its latency, the number of episodes of REM and their respective duration will be REM sleep macrostructure variables through which hyperarousal will be revealed. Since it was suggested that the proportion of REM sleep was positively related to subjective wakefulness in INS (Feige et al., 2008), PARA-I will have a higher proportion of REM sleep than PSY-I and GS. Consequently, PARA-I will have a shorter latency to REM sleep and more episodes of this sleep stage, which should be longer, compared to PSY-I and GS. We also predict that PSY-I will be spending less time in REM sleep than GS, confirming previous results (Bonnet and Arand, 1995; Feige et al., 2008; Jurysta et al., 2009; Merica et al., 1998; Nissen et al., 2011; Voderholzer et al., 2003). Compared to GS, PSY-I's REM sleep latency will be increased and they will have fewer episodes of REM sleep which should be shorter.

1.1.2. Microstructure

We hypothesize that hyperarousal in INS will be reflected through PSY-I and PARA-I's REM sleep microstructure. Therefore, we predict that both groups of INS will have more wake intrusions and arousals during REM sleep than GS. As for eye movements, because others have observed lower EMs in INS (Feige et al., 2008) and because PSY-I shall spend less time in this sleep stage than PARA-I and GS, we suggest lower EMs and density of eye movements (DEMs) in PSY-I than in the other two groups.

Finally, this study also aims at broadening our understanding on the relationship between REM sleep and the subjective perception of the quality and quantity of sleep in insomnia. We suggest that significant relationships will exist between subjective sleep evaluation and REM sleep for PSY-I and PARA-I, but not for GS. Since PARA-I tend to misperceive sleep, we predict that REM sleep should be positively related to subjective total wake time (TWT) and negatively correlated with subjective sleep efficiency (SE) and total sleep time (TST), confirming that REM sleep contributes to subjective wake time in insomnia (Feige et al., 2008). As for PSY-I, the relationships should be similar to those found for PARA-I, but of lower magnitude.

2. Material and methods

2.1. Participants

Participants were divided into three groups: 39 PSY-I, 27 PARA-I and 47 GS. All participants were aged between 25 and 55. Note that data from a proportion of these participants have been previously published elsewhere, in the context of ERP studies (77%: Bastien et al., 2013; 69%: Turcotte et al., 2011; 27%: Turcotte and Bastien, 2009), PSA studies (59%: St-Jean et al., 2013; 44%: St-Jean et al., 2012) and a napping study (43%: Péresse et al., 2013). In the present study, to be included in the PSY-I group, participants had to meet the following criteria: a) a

subjective complaint of insomnia characterized by difficulties initiating and/or maintaining sleep; b) insomnia must have been present at least three nights a week for more than six months; c) a complaint of at least one daytime consequence attributed to insomnia; d) distress or significant difficulties in social and/or occupational functioning, and e) a subjective sleep efficiency (SE) below 85% in their two week sleep diary prior to PSG recordings. Participants in the PARA-I group had to meet the same inclusion criteria as those in the PSY-I group, but their objective SE had to be superior to 85% and their total sleep time (TST) had to exceed 390 min during nights 2 and 3. An important discrepancy also had to be present between subjective and objective sleep variables using the same 2 nights; TST (≥ 60 minute discrepancy) and SE ($\geq 15\%$ discrepancy). For this study, GS had to report sleeping a minimum of 7 h per night, satisfaction with their sleep and no subjective sleep complaints. In addition to not meeting insomnia criteria, GS had to report a subjective SE $\geq 85\%$ in their sleep diaries.

Exclusion criteria for all participants were: a) a significant medical disorder; b) a major psychopathology; c) other sleep disorders such as sleep apnea (apnea–hypopnea > 15) or periodic limb movements during sleep (myoclonic index with arousal > 15); d) a strong dependency to tobacco; e) an ongoing psychological treatment; f) use of a medication known to affect sleep; g) a score > 23 on the *Beck Depression Inventory* (BDI; Beck et al., 1996); or h) a score > 15 on the *Beck Anxiety Inventory* (BAI; Beck and Steer, 1993). These criteria were consistent with those of the *ICSD-II* (AASM, 2005) and those of Bastien et al. (2008).

2.2. Procedure

All participants were recruited through media advertisements. Following a brief telephone screening interview, eligible participants were sent a set of questionnaires to evaluate psychological symptoms (BAI and BDI; Beck and Steer, 1993; Beck et al., 1996) and sleep difficulties [*Insomnia severity index* (ISI; Morin, 1993) and two weeks of sleep diaries (Morin, 1993)]. Those who met the inclusion criteria for any of the three groups were invited to the sleep laboratory for clinical interviews. Upon arrival, informed consent was obtained. The *Structured Clinical Interview for DSM-IV* (SCID-IV; Williams et al., 1992) was administered to rule out major psychopathologies and the *Insomnia Diagnosis Interview* (IDI; Morin, 1993) to explore the nature of insomnia symptoms. These evaluations were conducted respectively by a graduate student in a clinical psychology program (GST) and a sleep specialist (CHB). Participants meeting the study criteria underwent four consecutive nights of PSG recordings in the sleep laboratory. The first night was used for screening and adaptation and the fourth one for an ERP study. Clinical data of objective and subjective measures were collected during nights 2 and 3. We tried to respect participants' sleep schedule as much as possible, but participants had to be in bed by midnight and a minimum of 8 h of PSG was recorded every night. This procedure was approved by the ethics comity of the Centre de recherche de l'Institut universitaire en santé mentale de Québec (CER; # 183).

2.3. Measures

To evaluate psychological symptoms, the BAI (Beck and Steer, 1993), the BDI (Beck et al., 1996) and the SCID-IV (Williams et al., 1992) were administered. To portray sleep difficulties, the ISI (Morin, 1993) was completed as well as a two-week sleep diary which assesses subjective sleep quality, requiring participants to report their sleep habits, such as the number of awakenings, the length of each awakening, and the time spent in bed (Morin, 1993; Morin et al., 2011). Adequate psychometric properties have been reported for both questionnaires in previous studies (Bastien et al., 2001). Also, the IDI (Morin, 1993) was used to evaluate the presence of insomnia and its contributing factors.

2.3.1. PSG recordings

PSG was recorded during four consecutive nights. A standard PSG montage was used, including electroencephalography (EEG; F3, F4, Fz, C3, C4, Cz, P3, P4, Pz, O1, O2), electromyography (EMG; electrodes on chin), electrocardiography (ECG; electrode on heart) and electro-oculography (EOG; one electrode on the supra-orbital ridge of the right eye and another on the infra-orbital ridge of the left eye) recordings. Reference electrodes were fixed on the mastoids and the ground was on the forehead. On the first night, leg EMG (electrodes on the tibialis) and breathing devices (nasal flux to measure oxygen saturation and thoracic bands) were used in order to detect breathing disorders and limb movements. The inter-electrode impedance was maintained below 5 k Ω . To amplify the signal from the electrodes, a Grass Model 15A54 amplifier system (Astro-Med Inc., West Wrawick, USA; gain 10,000; bandpass 0.3–100 Hz) was used and PSG signals were digitized at a sampling rate of 512 Hz with the commercial product Harmonie (Stellate system, Montreal, Canada). PSG recordings were visually scored (Luna, Stellate system, Montreal, Canada) by experienced sleep technicians using Rechtschaffen and Kales' criteria (1968) at 20-second epochs.

In the present study, nights 2 and 3 were used to collect the variables of interest for REM sleep macrostructure which are: latency to REM sleep (REML) defined as time from the first epoch of stage 2 sleep to the first epoch of REM sleep; proportion of REM during the night which corresponded to the ratio of the total duration of REM sleep (REMD) over the total sleep time (TST) multiplied by 100; the number of periods of REM sleep where a period was defined as at least three consecutive REM sleep epochs separated by no more than two consecutive epochs of another stage. When there were three epochs or more of another stage between epochs of REM, another period was counted and the duration of these periods was also calculated. On the other hand, variables of interest for REM sleep microstructure are: number of eye movements (EMs) which were scored manually using Smith and Lapp's criteria (1991; i.e. a deviation in the EOG amplitude ≥ 25 μ V lasting for less than 2 s); density of eye movements (DEMs) corresponded to the ratio of the total EMs over the duration of REM sleep in minutes; arousals and wake intrusions during REM sleep were manually scored using The Atlas Task Force's criteria (1992) and ratio of arousals was calculated by adding the number of wake intrusions and arousals and dividing the total by the minutes spent in REM sleep, multiplying this score by 100.

2.4. Statistical analyses

One-way ANOVAs were ran to compare groups on socio-demographic data, psychological characteristics and sleep diary parameters. Independent samples *t*-tests were then performed on significant main effects. Since a between-group age difference was observed, which was correlated with some variables of interest, age was used as a covariate in subsequent analyses. Also, since no night effects were found, statistical analyses were computed using means of both experimental nights for each variables of interest.

Multivariate ANCOVAs were performed to compare groups on objective and subjective sleep parameters. Bonferroni corrections were then applied on significant main effects of groups. In order to compare participants on REM sleep macrostructure variables (REML, duration and proportion of REM sleep, number of episodes and duration of the first five episodes) and REM sleep microstructure variables (EMs, DEMs, wake intrusions, arousals and ratio of arousals), multivariate ANCOVAs were used and Bonferroni post hoc analyses were conducted on significant main effects.

Finally, bilateral Pearson's correlations were conducted between subjective sleep parameters and REM sleep duration and proportion using means of both nights. These analyses were computed in order to identify a possible contribution of REM sleep to the perception of a

bad night sleep often observed in PARA-I. Significance level was set at 0.05.

3. Results

3.1. Socio-demographic, psychological variables and sleep diaries parameters

Statistical analyses revealed no significant difference between PSY-I, PARA-I and GS for gender ($p = .06$) and education ($p = .90$) which varied from 6 to 25 years. PSY-I were significantly older than GS ($p = .02$), age varying between 25 and 55. No significant differences were observed between PSY-I and PARA-I for the reported duration of insomnia, ranging from 1 to 31 years. Analyses also showed that the severity of insomnia symptoms measured by the ISI varied between 0 and 10 and was significantly greater in PSY-I and PARA-I compared to GS ($p < .001$) and insomnia symptoms were qualified as more severe by PARA-I than PSY-I ($p = .004$). Both groups of INS reported more depressive symptoms (BDI; $p < .001$), scores ranging from 0 to 20, and anxious symptoms (BAI; $p < .001$), scores ranging from 0 to 15. For subjective sleep parameters from sleep diaries, all three groups were significantly different on SOL, WASO, TST and SE ($p > .001$). Table 1 illustrates means and SDs for each of the above variables.

3.2. Objective and subjective sleep parameters

First, statistical analyses conducted on objective sleep parameters revealed significant between group differences for TWT ($p = .02$), wake after sleep onset (WASO; $p = .05$) and SE ($p = .04$), PSY-I spending more time awake than GS (TWT and WASO), and having a smaller SE. PARA-I and the two other groups did not significantly differ on objective sleep parameters and there were no significant between group differences ($.19 \geq p \geq .30$) on objective sleep onset latency (SOL) and TST.

Second, statistical analyses conducted on subjective sleep parameters showed that groups were significantly different on SOL ($p < .001$), both groups of INS reporting longer time to fall asleep than GS ($p \leq .03$) and PARA-I having a longer SOL than PSY-I ($p \leq .003$). WASO was significantly greater in both groups of INS compared to GS ($p \leq .05$) and in PARA-I compared to PSY-I ($p < .001$). TWT was also significantly greater in PSY-I and PARA-I than GS ($p \leq .001$), and PARA-I reported spending more time awake during the night ($p < .001$) than PSY-I. Both groups of INS reported a shorter TST than GS ($p \leq .004$) and PARA-I spent less time asleep

than PSY-I ($p < .001$). Finally, SE was significantly poorer in PSY-I and PARA-I compared to GS ($p < .001$) and in PARA-I compared to PSY-I ($p < .001$). Refer to Table 2 for more details on objective and subjective sleep parameters.

3.3. REM sleep macrostructure

Statistical analyses conducted on REM sleep macrostructure variables revealed no significant between group differences for REML (ranging from 33 to 284.33 min), REMD (from 21 to 169.33 min), the proportion of REM sleep (8.70 to 40.67%), the number of episodes of this stage (from 2 to 11 episodes) and the duration of the first five REM sleep episodes.

3.4. REM sleep microstructure

Values of EMs varied from 12 to 1195 and from .18 to 11.04 for DEMs. ANCOVAs revealed no significant differences between groups for neither EMs nor DEMs ($p \leq .83$). Results nonetheless showed that PSY-I had significantly more wake intrusions during REM sleep than PARA-I ($p = .03$), ranging from 0 to 16, independently of grouping. Groups did not significantly differ ($.13 \geq p \geq .36$) on the other variables (REM sleep arousals and ratio of arousals). Arousals ranged from 2 to 54 and the proportion of REM sleep arousals varied from 3 to 64%. See Table 3 for detailed results.

3.5. Relationships between subjective sleep parameters and REM sleep

Independently of grouping, bilateral Pearson's correlations revealed relationships between REMD and subjective SE ($R = .35$, $p \leq .001$), TST ($R = .45$, $p \leq .001$) and TWT ($R = -.29$, $p = .002$). Also, the proportion of REM sleep was significantly associated with subjective SE ($R = .19$, $p = .04$) and marginally with subjective TST ($R = .18$, $p = .06$), but not with TWT ($R = -.14$, $p = .15$).

3.5.1. PARA-I

Analyses conducted on PARA-I showed that REMD was correlated with subjective SE ($R = .59$, $p = .001$), TST ($R = .59$, $p = .001$) and TWT ($R = -.49$, $p = .01$). For the proportion of REM sleep, significant associations were observed with subjective SE ($R = .504$, $p = .01$) and TST ($R = .49$, $p = .01$) and it was marginally linked with TWT ($R = -.38$, $p = .06$).

Table 1
Means (SD) of socio-demographic, psychological data and sleep diaries of psychophysiological insomnia sufferers (PSY-I), paradoxical insomnia sufferers (PARA-I) and good sleepers (GS).

	PSY-I <i>n</i> = 39	PARA-I <i>n</i> = 27	GS <i>n</i> = 47	F	p
Gender				2.84	.06
Female	21	21	24		
Male	18	6	23		
Age (years)	40.33 (9.05)	39.26 (9.10)	35.19 (8.96) ^a	3.84	.02*
Education (years)	15.37 (3.44)	15.62 (3.29)	15.70 (3.47)	0.10	.90
Insomnia duration (years)	12.37 (9.59)	9.38 (6.83)	—	<i>t</i> = 1.38	.17
Questionnaires					
ISI (severity score)	6.29 (1.69)	7.52 (1.33) ^a	1.06 (1.31) ^{a, b}	212.64	<.001**
BDI	7.53 (4.99)	6.95 (3.98)	2.83 (3.46) ^{a, b}	15.36	<.001**
BAI	5.97 (4.23)	5.90 (3.82)	2.39 (2.70) ^{a, b}	12.65	<.001**
Sleep diaries					
SOL	28.56 (21.87)	43.98 (30.10) ^a	10.18 (7.93) ^{a, b}	25.30	<.001**
WASO	46.01 (33.58)	63.94 (43.87) ^a	6.25 (7.62) ^{a, b}	37.98	<.001**
TST	387.48 (46.65)	309.19 (70.83) ^a	456.99 (39.53) ^{a, b}	72.53	<.001**
SE	78.25 (7.36)	64.05 (14.68) ^a	93.83 (3.49) ^{a, b}	103.67	<.001**

Insomnia severity index (ISI); Beck Depression Inventory (BDI); Beck Anxiety Inventory (BAI); sleep onset latency (SOL); wake after sleep onset; total sleep time (TST); sleep efficiency (SE).

^a Significant difference with PSY-I.

^b Significant difference with PARA-I.

* $p \leq .05$.

** $p \leq .001$.

Table 2

Means (SD) of objective and subjective sleep parameters of psychophysiological insomnia sufferers (PSY-I), paradoxical insomnia sufferers (PARA-I) and good sleepers (GS).

	PSY-I n = 39	PARA-I n = 27	GS n = 47	F	p
<i>Objective sleep parameters (mean nights 2 & 3)</i>					
SOL	13.80 (15.01)	11.57 (15.08)	9.55 (9.83)	1.72	.19
WASO	49.75 (38.34)	42.95 (26.10)	27.35 (23.60) ^a	3.07	.05*
TST	402.63 (51.19)	410.30 (29.91)	426.50 (42.33)	1.23	.30
TWT	63.55 (43.91)	54.51 (29.42)	36.90 (27.83) ^a	3.84	.02*
SE (%)	85.62 (9.07)	87.53 (5.78)	90.80 (5.87) ^a	3.23	.04*
<i>Subjective sleep parameters (mean nights 2 & 3)</i>					
SOL	26.14 (23.49)	44.98 (28.46) ^a	13.69 (14.03) ^{a,b}	17.27	<.001**
WASO	53.51 (41.04)	136.15 (83.99) ^a	19.91 (24.10) ^{a,b}	44.68	<.001**
TST	399.08 (59.60)	308.35 (60.26) ^a	447.90 (47.82) ^{a,b}	52.63	<.001**
TWT	79.26 (48.18)	171.69 (64.53) ^a	33.48 (31.49) ^{a,b}	70.40	<.001**
SE (%)	83.62 (10.01)	64.74 (11.99) ^a	93.00 (6.75) ^{a,b}	74.24	<.001**

Sleep onset latency (SOL); wake after sleep onset (WASO); total sleep time (TST); Total wake time (TWT); sleep efficiency (SE).

^a Significant difference with PSY-I.

^b Significant difference with PARA-I.

* p ≤ 0.05.

** p ≤ 0.001.

3.5.2. PSY-I

REMD was positively correlated with subjective SE ($R = .34$, $p = .03$) and TST ($R = .48$, $p = .002$). No significant relationships were found between the proportion of REM and subjective sleep parameters for PSY-I.

3.5.3. GS

As for GS, REMD was linked with SE ($R = .30$, $p = .04$) and TST ($R = .52$, $p \leq .001$), but the proportion of REM and subjective sleep measures were not correlated.

Table 3

Means (SD) of REM sleep macrostructure and microstructure variables of psychophysiological insomnia sufferers (PSY-I), paradoxical insomnia sufferers (PARA-I) and good sleepers (GS).

	PSY-I n = 39	PARA-I n = 27	GS n = 47	F	p
<i>REM sleep macrostructure variables (mean nights 2 & 3)</i>					
REML	84.96 (41.95)	82.17 (36.10)	82.52 (31.34)	0.04	.96
REMD	103.34 (27.25)	101.81 (18.63)	107.43 (17.18)	0.25	.78
Ratio REM (%)	25.06 (5.13)	24.58 (4.09)	25.24 (3.26)	0.22	.81
#Periods	5.28 (1.52)	4.83 (0.98)	5.26 (1.42)	0.85	.43
REM1	14.66 (6.18)	17.68 (8.71)	17.62 (11.34)	0.66	.52
REM2	19.83 (9.39)	18.31 (7.02)	19.42 (9.77)	0.43	.65
REM3	19.58 (12.13)	19.29 (9.57)	20.84 (9.42)	0.07	.93
REM4	23.28 (13.36)	22.70 (12.96)	23.37 (12.99)	0.28	.76
REM5	21.98 (11.99)	19.93 (14.74)	21.53 (12.25)	0.46	.64
<i>REM sleep microstructure variables (mean nights 2 & 3)</i>					
EMs	429.98 (204.20)	438.72 (279.53)	481.09 (185.28)	0.19	.83
DEMs	4.22 (1.86)	4.44 (2.52)	4.48 (1.47)	0.11	.90
WI	3.60 (2.15)	2.25 (1.35) ^a	2.64 (1.53)	3.69	.03*
AR	15.67 (9.30)	12.67 (6.26)	15.93 (7.51)	1.04	.36
Ratio arousals (%)	19.55 (9.62)	14.64 (5.56)	17.71 (7.10)	2.13	.13

Latency to REM sleep (REML); Duration of REM sleep (REMD); Proportion of REM sleep (Ratio REM); Number of periods of REM sleep (#Periods); Duration of each period of REM sleep (REM1; REM2; REM3; REM4; REM5); Number of eye movements (EMs); Density of eye movements (DEMs); Wake intrusions during REM sleep (WI); REM sleep arousals (AR); Proportion of REM sleep arousals (Ratio arousals).

^a Significant difference with PSY-I.

^b Significant difference with PARA-I.

* p ≤ 0.05.

** p ≤ 0.001.

4. Discussion

In the present study, GS and INS, classified in psychophysiological and paradoxical types, were compared on REM sleep macrostructure and microstructure variables in order to determine if REM sleep could be a potential indicator of hyperarousal in insomnia. Socio-demographic data revealed that PSY-I were significantly older than GS. Since age has an impact on sleep parameters (Arbus and Cochen, 2010; Ohayon et al., 2004; Pace-Schott and Spencer, 2011), age was used as a covariate in subsequent analyses.

In order to determine if laboratory nights were representative of the usual sleep patterns of PSY-I and PARA-I, groups were compared on means of objective and subjective sleep parameters of both nights of PSG recordings. PSY-I had significantly longer TWT and WASO as well as a poorer SE compared to GS. These observations correspond to the usual sleep pattern of prolonged nocturnal awakenings characterizing PSY-I, which consequently impacted sleep efficiency. A prolonged time in bed in PSY-I might explain the absence of significant difference between PSY-I and GS for TST. However, their poor sleep pattern was translated through SE value, which is a far more meaningful indicator of sleep quality. As for SOL, the lack of significant difference between PSY-I and GS might result from the type of nocturnal difficulties experienced by PSY-I. In fact, participants in the PSY-I group suffered mainly of middle and/or terminal insomnia rather than initial insomnia. Thus, SOL was similar between groups and sleep initiation difficulty was not PSY-I's main concern. As for PARA-I, they were similar to the other two groups on objective sleep parameters (SOL, WASO, TST, TWT and SE). On the other hand, subjective sleep parameters (SOL, WASO, TST, TWT and SE) illustrate important discrepancies with objective measures in PARA-I. They tend to grossly underestimate sleep quality (SOL, WASO, TWT and SE) and quantity (TST), confirming sleep misperception, which is the main attribute of their diagnosis. Overall, objective and subjective night parameters confirmed that nights spent in the laboratory seemed to be representative of PSY-I and PARA-I's usual sleep patterns.

The first objective of this study was to determine if REM sleep macrostructure was a potential indicator of hyperarousal in insomnia. We hypothesized that REM sleep macrostructure would reflect hyperarousal, especially in PARA-I. Contrary to our expectations, REM sleep macrostructure does not seem to be an adequate indicator of hyperarousal neither in PARA-I nor in PSY-I. In fact, as in Riemann et al.'s study (2012), our investigation failed to find shortened REML in INS compared to GS. Also, the absence of significant differences between the three groups on REMD and the proportion of REM sleep suggests that the macrostructure of REM might not be an adequate indicator of hyperarousal in insomnia. This observation is surprising and difficult to reconcile with previous literature which found a smaller amount of REM sleep in INS compared to GS (Bonnet and Arand, 1995; Feige et al., 2008; Jurysta et al., 2009; Merica et al., 1998; Nissen et al., 2011; Voderholzer et al., 2003).

Discrepancies between our and those previous studies might reside in the set of criteria used for defining insomnia [Sleep difficulties (SOL ≥ 45 min and/or WASO ≥ 60 min) at least four nights/week for more than a year (Bonnet and Arand, 1995); sleep difficulties lasting for more than a month (Jurysta et al., 2009)], which were not always clearly stated either (Nissen et al., 2011). Additionally, previous results were based on one experimental night only, failing to capture the night-to-night variability characterizing INS, thus compromising the generalization of these observations (Vallièrès et al., 2011). Also, since age has an important impact on sleep parameters (Arbus & Cochen, 2010; Ohayon et al., 2004; Pace-Schott and Spencer, 2011), the large age range of participants might have increased the severity of sleep difficulties in INS in previous studies (Feige et al., 2008; Jurysta et al., 2009; Voderholzer et al., 2003). Lastly, in the present study, differences between the mean objective SE of GS (90.8%) vs. PSY-I (85.6%) and PARA-I (87.5%) were much smaller than previous results [SE of GS (94%) vs. INS (75%; Bonnet and Arand, 1995); SE of GS (81%) vs. INS

(70%; Nissen et al., 2011)]. We can suppose that the greater is the discrepancy between SE of groups of sleepers, and the higher is the chance of observing differences in REM sleep macrostructure. In fact, a 'good' SE should increase the opportunity of REM sleep and a 'poor' SE should lead to a decreased opportunity of having REM sleep. In sum, the small difference between groups on objective SE could explain the absence of significant differences between GS, PSY-I and PARA-I for the amount of REM sleep in our investigation. Although differences in REM sleep macrostructure of INS compared to GS were observed in the majority of previous studies, our results are similar to those of Hairston et al. (2010).

Finally, the number of REM sleep periods and their duration were similar between groups. These results fail to confirm previous observations of shorter REM sleep periods in INS (Merica et al., 1998). A smaller sample size (20 INS and 19 GS) and only one night of clinical data might explain these contradictory results. In fact, limiting experimental designs to one night makes it difficult to capture the night-to-night variability often observed in INS and questions the representativeness of obtained observations. Because of our large sample size (39 PSY-I, 27 PARA-I and 47 GS) and clinical sleep data derived from two sleep nights, we believe our results might therefore be more representative of the general population and thus, more generalizable.

The second objective of the present study was to evaluate REM sleep microstructure as a potential indicator of hyperarousal in insomnia. We hypothesized that REM sleep microstructure would reflect hyperarousal in PSY-I and PARA-I. Our initial hypothesis is partially confirmed since wake intrusions in REM appeared to reflect hyperarousal in PSY-I. First, EMs and DEMs do not appear as adequate indicators of hyperarousal neither in PARA-I nor in PSY-I. Contrary to Feige et al. (2008), our groups were similar on EMs and DEMs, implying that EMs during REM sleep are not related to hyperarousal. Discrepancies between our study and Feige et al. (2008) might reside in the use of a different set of criteria to define EMs. In our investigation, we defined EMs as a deviation in EOG amplitude of more than 25 μ V based on Smith and Lapp's criteria (1991), whereas Feige et al. (2008) used 70 μ V. Differences in EMs between INS and GS might therefore reside in EMs of larger amplitudes. Grouping EMs in function of their amplitude in further investigations would help determining where differences between groups of sleepers reside, thus leading to a better understanding of insomnia.

As expected, PSY-I displayed more wake intrusions in REM sleep than PARA-I, reflecting a fragmented sleep and corresponding to the ICSD-II diagnostic criteria (AASM, 2005). In PSY-I, hyperarousal might be reflected through elevated number of wake intrusions in REM, identifying this variable as a potential indicator of heightened arousal. Thus far, REM sleep intrusions (wake and arousals) were studied by only one group, Feige et al. (2008). These authors observed an increased number of wake intrusions/arousals during REM sleep in INS compared to GS. Our results are in line with theirs, except that we did not find significant between group differences for arousals. Methodologically, the two studies differ. For example, results in Feige and colleagues' study (2008) were derived from one experimental night. Also, fixed bedtimes and rise times for every participant was used in their study and finally, the age range was much larger than in our study (17 to 79 years and 25 to 55 years respectively). Because of limited available data, more investigations addressing these methodological issues to uncover the expression of hyperarousal in REM sleep are needed. Further studies should limit the age range of recruited participants and collect clinical data over few (more than 2) PSG recording nights to better capture INS sleep patterns.

The overall absence of significant between group differences in the macrostructure and microstructure of REM sleep suggests that their adequacy as indicators of hyperarousal in insomnia is limited. Perhaps, finer REM sleep microstructure variables such as the density of the activities in the different frequency bands measured using PSA might be a better reflection of this heightened arousal in insomnia. Limited literature exists on this subject, but so far, results obtained using PSA tend to show significant differences between PSY-I, PARA-I and GS in the

density of each frequency bands during REM sleep (Freedman, 1986; Krystal et al., 2002; Merica et al., 1998; Perlis et al., 2001; St-Jean et al., 2012, 2013). Therefore, it seems that this finer aspect of REM sleep microstructure is somehow related to the hyperarousal typically suggested in INS.

In order to broaden our understanding of insomnia, the last objective of this study was to establish the relationship between REM sleep and subjective perception of sleep quality and quantity. We postulated that subjective sleep parameters (SE, TST and TWT) will be correlated with REM sleep duration and proportion for PSY-I and PARA-I, but not for GS. This hypothesis is only partially confirmed. Interestingly, obtained results are opposite from our initial predictions. As such, the more time was spent in REM sleep, the better was perceived sleep quality and quantity (SE and TST); these relationships being stronger in PARA-I than in the two other groups of sleepers. Similar results were observed between REMD and subjective TWT, but only for PARA-I, showing that the more time they spent in REM sleep, the less they were prone to estimate their sleep as wake.

These results are surprising as they suggest that greater objective quantity of REM sleep contributes to a better perception of sleep quality and quantity, especially for PARA-I. Results are also difficult to reconcile with previous literature. In fact, Feige and colleagues (2008) observed that a greater amount of REM sleep contributed to a greater subjective feeling of being awake in insomnia individuals. These authors suggested that this observation might be explained by the fact that this sleep stage is electroencephalographically similar to wake. Alternatively, it is possible that more intense and vivid dream imagery activity generally occurring in REM sleep might contribute to these relationships between the duration of REM sleep and subjective sleep parameters. Independently of grouping, a higher time spent in REM sleep will increase the opportunity of having intense and vivid dream imagery. Since these dreams are bizarre in nature and thus more easily remembered, sleepers are more likely to realize that they were asleep when dreams occurred and therefore, less prone to misperceive REM sleep as wake. Consequently, REM sleep imagery might explain that an increased duration of REM sleep lead to better perceived sleep quality and quantity in PSY-I, PARA-I and GS. This hypothesis remains to be tested.

Interestingly, correlations established between the proportion of REM sleep and subjective sleep measures were significant for SE and TST and marginally significant for TWT, in PARA-I only. The higher was the proportion of REM sleep, the better PARA-I perceived their sleep, which might again be linked to dream activity, considering that more vivid and intense dream imagery occurs in REM sleep. PARA-I might experience increased dream activity compared to PSY-I and GS during REM sleep. Although PARA-I's dreams might contribute to a good subjective evaluation of sleep, dreams' characteristics may also be indicators of hyperarousal. It is possible that PARA-I knew they were asleep during the night because they dreamt and they could remember their dreams in the morning. However, intense dream activity might contribute to an agitation of PARA-I's sleep and thus may partly explain feelings of restlessness during the day. REM sleep dream imagery activity might be a promising research avenue in insomnia. Further investigations on dream recall frequency and dream content are needed to determine if these REM sleep variables could be good indicators of hyperarousal in different types of insomnia.

There are some limitations that can be pointed out in this study. It is possible that two experimental nights were not enough to capture the night-to-night variability proper to insomnia, even though two nights are more than what is generally used in insomnia research. Also, nights spent in the laboratory might influence REM sleep and therefore not be representative of the usual nights of GS and INS. At-home ambulatory devices should be used in further investigations in order to limit the possible impacts of the laboratory settings on REM sleep. This type of protocol would help portraying GS and INS' sleep.

To conclude, REM sleep macrostructure, as studied here, is limited for reflecting hyperarousal in insomnia. However, increased wake

intrusions in REM sleep in PSY-I are an aspect of the microstructure that might reflect heightened arousal. Nonetheless, more studies need to be conducted in order to identify variables which could contribute to hyperarousal and also help at differentiating insomnia types. Further investigations on dream imagery activity in insomnia may be promising avenues of research in order to increase our understanding of hyperarousal in insomnia. The exploration of dreams combined with finer EEG analyses such as PSA might also provide interesting cues to further our identification of hyperarousal during sleep.

Acknowledgments

We would like to thank Sonia Petit for analysing PSG recordings and all the research assistants who helped in data entry. The present study was also made possible by funds from the Canadian Institutes of Health Research to CHB (49500 and 86571).

References

- American Academy of Sleep Medicine, 2005. *International Classification of Sleep Disorders*. 2nd edn. American Academy of Sleep Medicine, Chicago (401 pp.).
- American Academy of Sleep Medicine, 2014. *International Classification of Sleep Disorders*. 3rd edn. American Academy of Sleep Medicine, Illinois.
- American Psychiatric Association, 2013. *Diagnostic and Statistical Manual of Mental Disorders*. 5th edn. American Psychiatric Publishing, Arlington (991 pp.).
- Arbus, C., Cohen, V., 2010. Les modifications du sommeil avec l'âge. *Psychol. Neuropsychiatr. Vieil.* 8, 7–14.
- Baglioni, C., Regen, W., Teghen, A., Spiegelhalter, K., Feige, B., Nissen, C., Riemann, D., 2013. Sleep changes in the disorder of insomnia: a meta-analysis of polysomnographic studies. *Sleep Med. Rev.* <http://dx.doi.org/10.1016/j.smrv.2013.04.001>.
- Bastien, C.H., 2011. Insomnia: neurophysiological and neuropsychological approaches. *Neuropsychol. Rev.* 21, 22–40.
- Bastien, C.H., Vallières, A., Morin, C.M., 2001. Validation of the insomnia severity index as an outcome measure for insomnia research. *Sleep Med.* 2, 297–307.
- Bastien, C.H., LeBlanc, M., Carrier, J., Morin, C.H., 2003. Sleep EEG power spectra, insomnia, and chronic use of benzodiazepines. *Sleep* 26, 313–317.
- Bastien, C.H., Guimond, S., St-Jean, G., Lemelin, S., 2008. Signs of insomnia in borderline personality disorder individuals. *J. Clin. Sleep Med.* 4, 462–470.
- Bastien, C.H., Turcotte, I., St-Jean, G., Morin, C.M., Carrier, J., 2013. Information processing varies between insomnia types: measures of N1 and P2 during the night. *Behav. Sleep Med.* 11, 56–72.
- Beck, A.T., Steer, R.A., 1993. *Beck Anxiety Inventory Manual*. Psychological Corporation, San Antonio.
- Beck, A.T., Steer, R.A., Brown, G.K., 1996. *Manual for Beck Depression Inventory II (BDI-II)*. Psychology Corporation, San Antonio.
- Bonnet, M.H., Arand, D.L., 1995. 24-hour metabolic rate in insomniacs and matched normal sleepers. *Sleep* 18, 581–588.
- Bonnet, M.H., Arand, D.L., 1998. The consequences of a week of insomnia II: patients with insomnia. *Sleep* 21, 359–368.
- Bonnet, M.H., Arand, D.L., 2000. Activity, arousal, and the MSLT in patients with insomnia. *Sleep* 23, 1–8.
- Edinger, J.D., Bonnet, M.H., Bootzin, R.R., et al., 2004. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep* 27, 1567–1596.
- Edinger, J.D., Means, M.K., Carney, C.E., Krystal, A.D., 2008. Psychomotor performance deficits and their relation to prior nights' sleep among individuals with primary insomnia. *Sleep* 31, 599–607.
- Espie, C.A., 2002. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorders in adults. *Annu. Rev. Psychol.* 53, 215–243.
- Feige, B., Al-Shajlawi, A., Nissen, C., et al., 2008. Does REM sleep contribute to subjective wake time in primary insomnia? A comparison of polysomnographic and subjective sleep in 100 patients. *J. Sleep Res.* 17, 180–190.
- Feige, B., Baglioni, C., Spiegelhalter, K., Hirscher, V., Nissen, C., Riemann, D., 2013. The microstructure of sleep in primary insomnia: an overview and extension. *Int. J. Psychophysiol.* <http://dx.doi.org/10.1016/j.jpsycho.2013.04.002>.
- Freedman, R., 1986. EEG power in sleep onset insomnia. *Electroencephalogr. Clin. Neurophysiol.* 63, 408–413.
- Hairston, I.S., Talbot, L.S., Eidelman, P., Gruber, J., Harvey, A.G., 2010. Sensory gating in primary insomnia. *Eur. J. Neurosci.* 31, 2112–2121.
- Harvey, A.G., 2002. A cognitive model of insomnia. *Behav. Res. Ther.* 40, 869–893.
- Jurysta, F., Lanquart, J.P., Sputaels, V., et al., 2009. The impact of chronic primary insomnia on the heart rate – EEG variability link. *Clin. Neurophysiol.* 20, 1054–1060.
- Krystal, A.D., Edinger, J.D., Wohlgemuth, W.K., Marsh, G.R., 2002. Non-REM sleep EEG frequency spectral correlates of sleep complaints in primary insomnia subtypes. *Sleep* 25, 630–640.
- Lamarche, C.H., Ogilvie, R.D., 1997. Electrophysiological changes during the sleep onset period of psychophysiological insomniacs, psychiatric insomniacs, and normal sleepers. *Sleep* 20, 724–733.
- Merica, H., Blois, R., Gaillard, J.M., 1998. Spectral characteristics of sleep EEG in chronic insomnia. *Eur. J. Neurosci.* 10, 1826–1834.
- Morin, C.M., 1993. *Insomnia: Psychological Assessment and Management*. Guilford Press, New York (238 pp.).
- Morin, C.M., LeBlanc, M., Bélanger, L., Ivers, H., Mérette, C., Savard, J., 2011. Prevalence of insomnia and its treatment in Canada. *Can. J. Psychiatry* 56, 540–548.
- Nissen, C., Kloepper, C., Feige, B., Piosczyk, H., Spiegelhalter, K., Voderholzer, U., Riemann, D., 2011. Sleep-related memory consolidation in primary insomnia. *J. Sleep Res.* 20, 129–136.
- Ohayon, M.M., 2002. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med. Rev.* 6, 97–111.
- Ohayon, M.M., Carskadon, M.A., Guilleminault, C., Vitiello, M.V., 2004. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. *Sleep* 27, 1255–1273.
- Okura, K., Lavigne, G.J., Huynh, N., Manzini, C., Fillipini, D., Montplaisir, J.Y., 2008. Comparison of sleep variables between chronic widespread musculoskeletal pain, insomnia, periodic leg movements syndrome and control subjects in a clinical sleep medicine practice. *Sleep Med.* 9, 352–361.
- Pace-Schott, E.F., Spencer, R.M., 2011. Age-related changes in the cognitive function of sleep. *Prog. Brain Res.* 191, 75–89.
- Parrino, L., Milioli, G., De Paolis, F., Grassi, A., Terzano, M.G., 2009. Paradoxical insomnia: the role of CAP and arousals in sleep misperception. *Sleep Med.* 10, 1139–1145.
- Perlis, M.L., Giles, D.E., Mendelson, W.B., Bootzin, R.R., Wyatt, J.K., 1997. Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *J. Sleep Res.* 6, 179–188.
- Perlis, M.L., Smith, M.T., Andrews, P., Orff, H., Giles, D.E., 2001. Beta/gamma activity in patients with insomnia and secondary insomnia and good sleepers controls. *Sleep* 24, 110–117.
- Pérusse, A.D., Turcotte, I., St-Jean, G., Ellis, J., Hudon, C., Bastien, C.H., 2013. Types of primary insomnia: is hyperarousal also present during napping? *J. Clin. Sleep Med.* 9, 1273–1280.
- Rechtschaffen, A., Kales, A., 1968. *A manual of Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects*. Brain Information Service/Brain Research Institute, University of California, Los Angeles.
- Riemann, D., Spiegelhalter, K., Nissen, C., Hirscher, V., Baglioni, C., Feige, B., 2012. REM sleep instability – a new pathway for insomnia? *Pharmacopsychiatry* 45, 167–176.
- Roehrs, T.A., Randall, S., Harris, E., Maan, R., Roth, T., 2011. MSLT in primary insomnia: stability and relation to nocturnal sleep. *Sleep* 34, 1647–1652.
- Smith, C., Lapp, L., 1991. Increases in number of REMS and REM density in humans following an intensive learning period. *Sleep* 14, 325–330.
- Stepanski, E., Zorick, F., Roehrs, T., Young, D., Roth, T., 1988. Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. *Sleep* 11, 54–60.
- St-Jean, G., Turcotte, I., Bastien, C.H., 2012. Cerebral asymmetry in insomnia sufferers. *Front. Neurol.* 3, 1–12.
- St-Jean, G., Turcotte, I., Pérusse, A.D., Bastien, C.H., 2013. REM and NREM spectral power analysis on two consecutive nights in psychophysiological and paradoxical insomnia sufferers. *Int. J. Psychophysiol.* 89, 181–194.
- Thacher, P.V., Pigeon, W.R., Perlis, M.L., 2006. Do patients with sleep maintenance insomnia have a problem with sleep maintenance? *Behav. Sleep Med.* 4, 203–218.
- The Atlas Task Force, 1992. EEG arousals: scoring rules and examples. *Sleep* 15, 173–184.
- Turcotte, I., Bastien, C.H., 2009. Is quality of sleep related to the N1 and P2 ERPs in chronic psychophysiological insomnia sufferers? *Int. J. Psychophysiol.* 72, 314–322.
- Turcotte, I., St-Jean, G., Bastien, C.H., 2011. Are individuals with paradoxical insomnia more hyperaroused than individuals with psychophysiological insomnia? Event-related potentials measures at the peri-onset of sleep. *Int. J. Psychophysiol.* 81, 177–190.
- Vallières, A., Ivers, H., Beaulieu-Bonneau, S., Morin, C.M., 2011. Predictability of sleep in patients with insomnia. *Sleep* 34, 609–617.
- Voderholzer, U., Al-Shajlawi, A., Weske, G., Feige, B., Riemann, D., 2003. Are there gender differences in objective and subjective sleep measures? A study of insomniacs and healthy controls. *Depress. Anxiety* 17, 162–172.
- Wicklow, A., Espie, C.A., 2000. Intrusive thoughts and their relationship to actigraphic measurement of sleep: toward a cognitive model of insomnia. *Behav. Res. Ther.* 38, 679–693.
- Williams, J.B.W., Gibbon, M., First, M.B., et al., 1992. The structured clinical interview for DSM-III (SCID). 2. Multisite test-retest reliability. *Arch. Gen. Psychiatry* 49, 630–636.